"ROLE OF ACARBOSE IN MANAGEMENT OF GESTATIONAL DIABETES MELLITUS"

Dissertation submitted to

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M.D. OBSTETRICS AND GYNAECOLOGY

BRANCH II

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MADRAS MEDICAL COLLEGE
CHENNAI

MARCH 2009

CERTIFICATE

This is to certify that this dissertation entitled **"“ROLE OF ACARBOSE IN MANAGEMENT OF GESTATIONAL DIABETES MELLITUS"** is a bonafide work done by **Dr. AMITHA INDERSEN,** postgraduate student in M.D (Obstetrics and Gynaecology) under my overall supervision and guidance at the Institute of Obstetrics and Gynaecology, Madras Medical College, Chennai in partial fulfillment of the regulations of Tamilnadu Dr. M.G.R. medical university for the award of M.D degree in Obstetrics and Gynaecology.

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ETHICAL COMMITTEE CERTIFICATE

DATED : 25/2/08

I, Dr. AMITHA INDERSEN apply for the ethical committee certificate for the project "**ROLE OF ACARBOSE IN MANAGEMENT OF GESTATIONAL DIABETES MELLITUS".** Under the guidance of Prof.Dr.Anjalakshi Chandrashekar M.D., D.G.O.,Ph.D.,Institute of Obstetrics and Gynaecology ,Egmore, Chennai-8.

I understand the implications of doing research with human subjects and will fully comply with the regulations and keep the dignity and protect the health of subjects at all costs.

SIGNATURE OF THE POSTGRADUATE STUDENT

I have no objection to guiding this postgraduate student in the project mentioned above.I Shall supervise to the extent that all the human rights are protected and research is carried on with utmost humanitarian principles.

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I Certify that this project has been presented in front of the Ethical Committee , duly formatted in this institution and that all the members of the ethical committee have given permission to conduct this research.

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CONTENTS

INTRODUCTION

Diabetes Mellitus is a clinical syndrome characterized by hyperglycemia due to absolute or relative deficiency of insulin. Lack of insulin, whether absolute or relative affects the metabolism of carbohydrate, protein, fat(1) .

Pregnancy is characterized by mild fasting hypoglycemia, post-prandial hyperglycemia, hyperinsulinism and insulin resistance- a diabetogenic stress. Normally pregnant woman elaborates an increased insulin production by 30% above her non pregnant state. A woman who is unable to achieve adequate insulinogenic compensation develops Gestational Diabetes. Pregnancy unmasks the minor intolerance of carbohydrate metabolism in subjects with reduced pancreatic islet cell reserve (2).

Gestational Diabetes was defined as carbohydrate intolerance of variable severity with its onset or first recognition during pregnancy. Use of this term was encouraged in order to communicate the need for increased surveillance and to convince the woman of the need for further testing postpartum(3)

Gestational Diabetes is often asymptomatic and associated with increased fetal and neonatal morbidity and mortality. Good glycemic control reduces the risk of complication(4).

CARBOHYDRATE METABOLISM IN NON DIABETIC PREGNANCY(5)

Factors contributing to insulin resistance

Production of placental somatomammotrophin, increased production of estrogen progesterone, increased insulin destruction by placental enzyme like insulinase.

Changes in Gluconeogenesis

Fetus continuously uses fuels from the mother. It uses alanine and other amino acids and depletes the mother of a major gluconeogenic source.

Increased Lipolysis

The mother uses fat for caloric needs and saves glucose for the fetus.

METABOLIC CHANGES DURING FASTING

During fasting there is decrease in plasma and amino acids. There is higher plasma concentration of free acids, triglycerides. During fasting for a longer period there is switch in metabolism from glucose to lipid which is termed "accelerated starvation" by Freinkel(8)

METABOLIC CHANGES DURING FED STATE(5)

During the first few hours, glucose absorbed from the gastro intestinal tract provides for the metabolic needs of brain and other organs. The absorbed glucose in excess of these needs is used to rebuild fuels in liver, muscle, fat and to provide supply of glucose for the fetus. This is facilitated anabolism.

REVIEW OF LITERATURE

HISTORY

Diabetes was described more than 2000 years ago. An ancient documentation by **Susruta** in India at about 400 B.C. has described the diabetic syndrome as characterized by a 'honeyed urine". The word Diabetes (to flow through) was coined by Greek physician **Aretus Or Cappadocia** in first century (150 A.D) from the word siphon (sweet taste). The word mellitus (honeyed) was added by **John Rollo** in 18th century.

In 1674, **Thomas Willis,** a Physician, Anatomist and a professor of Natural philosophy at Oxford discovered by tasting that the urine of diabetic persons was "wonderfully sweet as if imbued with honey or sugar". **Willis** could not explain the chemical nature of the sweet substance. It was **Mathew Dobson** of Manchester, England who in 1776 demonstrated that diabetics actually excrete sugar in urine. It was **John Rollo**, surgeon general of Royal artillery who first applied the discovery of glycosuria by Dobson to the quantitative metabolic study of diabetes.

It was **Claude Bernard** who studied the association between pancreas and Diabetes. The name Insulin was coined by **De Mayer** (1909). In 1921 **Fredrick Banting** and **Charles Best** with the help of Chemist **J.B.Collip** succeeded in fulfilling all of the criteria for the therapeutic active Insulin.

Glucose homeostasis reflects a precise balance between hepatic glucose production and peripheral glucose uptake and utilization. Insulin is the mediator of most signals and hormones resulting in integrated control of glucose supply and utilization. In

the fasting state low insulin levels increase glucose production by promoting hepatic gluconeogensis and glycogenolysis and reduce glucose uptake in insulin sensitive tissue. Glucagon also stimulates glycogenolysis and gluconeogenesis by the liver and renal medulla. Post prandially the glucose load elicits a rise in insulin and fall in glucagon. The major portion of post prandial glucose is utilized by skeletal muscles, an effect of insulin stimulated glucose uptake(6).

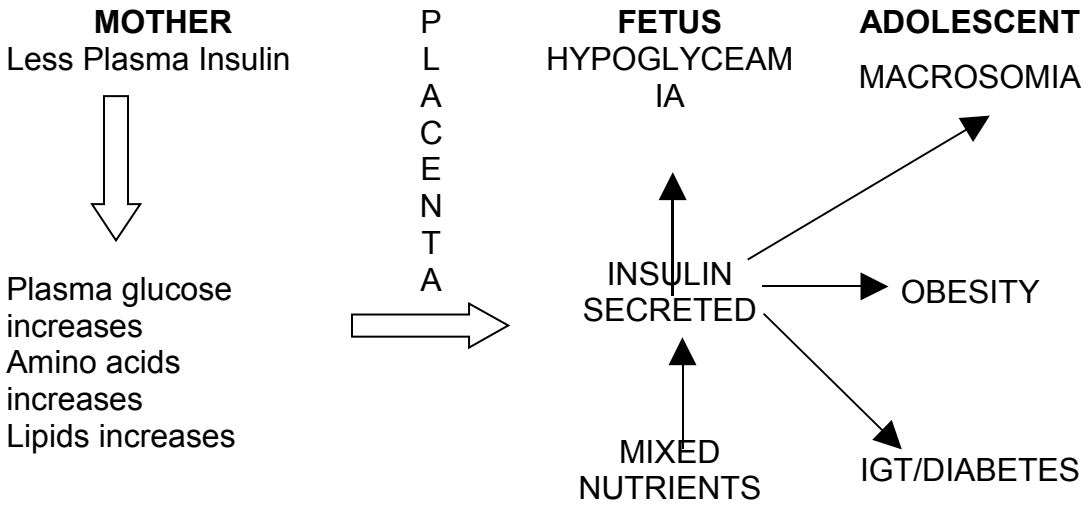
PATHOPHYSIOLOGY OF PERINATAL MORBIDITY

The **Pederson hypothesis** suggested that in diabetic pregnancy maternal hyperglycaemia is rapidly translated into fetal hyperglycaemia**.** The fetal pancreas responds to this glycaemic stimulatus with islet cell hypertrophy and hyperplasia and fetal hyperinsulinism results. It is the fetal hyperinsulinemia that results in diabetic fetopathy or prenatal morbidity seen in such pregnancies. According to modified Pederson hypothesis the non-glucose secretagogues for fetal pancreatic insulin also play a role in perinatal morbidity (25).

MODIFIED PEDERSEN / FRIENKEL'S HYPOTHESIS

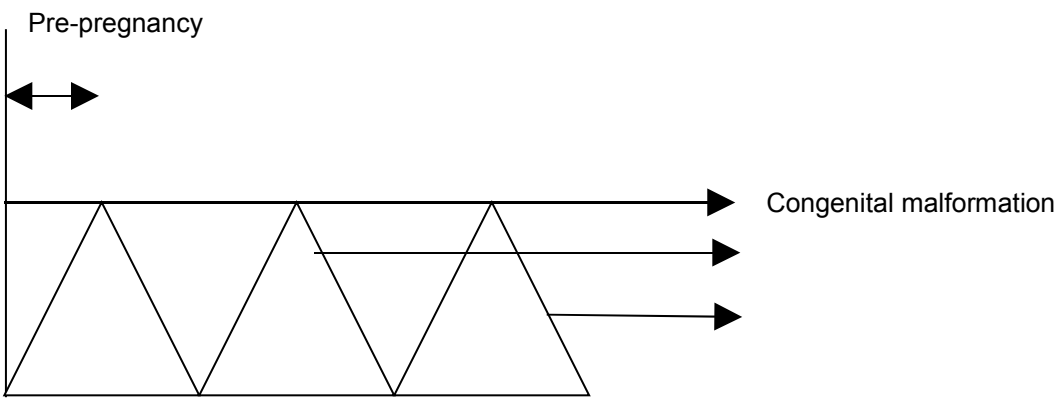
The transport of maternal fuel to the fetus requires normal placental intermediary metabolism and normal supply of substrates. Because diabetes may result in markedly abnormal concentrations of maternal glucose, fatty acids**,** triglycerides and amino acids these may get transported to the fetus. Unlike the fetus of early gestation, the fetus of the late gestation is well equipped to synthesise and replace insulin from its pancreas and protect itself against brunt of abnormal brunt of abnormal fuel mixture, thereby

normalizing blood sugar in circulation but in the process resulting in hyperinsulinemia of the fetus and due to its anabolic action causes macrosomia and associated complications.



Gestational carbohydrate intolerance is asymptomatic. The subjects destined to develop gestational diabetes mellitus have limited pancreatic insulin reserve and reduced insulin sensitivity. The stress of pregnancy due to counter insulin hormones overwhelms the insulin reserves. Hence in the plasma, levels of all classes of fuels - amino acids, fatty acids and glucose are elevated which are delivered to the fetus. This change is seen more during the latter half of pregnancy when counter insulin factors and insulin resistance is experienced. The fetal transportation of abnormal fuels result in hyperinsulinemia and during the delivery, RDS, neonatal hypoglycemia, hypocalcaemia, hyperbilirubinemia become the major concern for the subjects with gestational diabetes.

FREINKEL'S FUEL MEDIATED HYPOTHESIS:



Behavioural changes
Anthropometric changes

1st Trimester 2nd Trimester 3rd Trimester

Effects of abnormal glucose tolerance on the mother and the fetus:

On the mother:

•Pre-eclampsia (16) is seen in 13.7% in Gestational Diabetes Mellitus, and 14.1% to 27% in established diabetes mellitus. Pre - eclampsia and pregnancy induced hypertension are more common in patients with Gestational Diabetes than in controls(17). **Combs et al Rosenn et al**(18) reported a significant association between poor glycaemic control and pre - eclampsia or pregnancy induced hypertension.

•The incidence of chronic hypertension(19) is 2.5% among Gestational Diabetes, against 0.3% in the non diabetic control group.

•Overall incidence of ketoacidosis is 0.7% especially following beta agonist therapy. **Kilvert et al.,** reported one case of diabetic ketoacidosis in 150cases of Gestational Diabetes Mellitus(20). Diabetic ketoacidosis is preventable and the prevention can be accomplished with optimal glycemic control.

•The incidence of Hydramnios ranges from 2.0 to 2.1%(21) in gestational diabetes mellitus and it is about 20% to 30% in overt diabetics. Most infants of hydramniotic diabetic pregnancy are structurally normal, associated with increased incidence of preterm labour and premature rupture of membrane.

•Pyelonephritis was reported in 1.2% gestational diabetes mellitus and 3.6% of overt diabetes(19). There is no difference in the incidence of pyelonephritis in gestational

diabetes mellitus and control groups.

•Preterm labour complicated 8.1 of gestational diabetes mellitus and there is no significant difference in the preterm labour rate between gestational diabetes mellitus and control groups. There is significant correlation between preterm labour and uro-genital infection. (Candida and Trichomoniasis). **Molsted and Pedersen (22)** speculated that, hormonal differences increased the frequency of preterm labour in a diabetic than in non-diabetic women.

•Spontaneous pre-term delivery is one of the important contributions to perinatal mortality in diabetic pregnancies(22). In a Scandinavian report by **Molsted and Pederson,** the incidence of preterm labour with delivery was 14.6% in gestational diabetes mellitus versus 18 to 24% in established diabetes mellitus, and in the control group it was 12%.

•The incidence of primary caesarean section among gestational diabetes mellitus was 16.5% and 6% among the control groups, There is a higher total caesarean section rate in gestational diabetes mellitus than controls(24).

Fetal Problems associated with maternal hyperglycemia

First Trimester	Second Trimester	Third Trimester
Malformations	Hypertrophic cardiomyopathy	Hypoglycemia
Growth retardation	Polyhydramnios	Hypocalcemia
	Placental insufficiency	Hyperbilirubinemia
	Pre-eclampsia	Respiratory - distress Syndrome
	Fetal loss	Macrosomia
	Low IQ	Hypomagnesemia

		Intrauterine death

- Hypoglycemia is one of the common causes for perinatal morbidity. It is defined as blood sugar level less than 40mg% in any infant regardless of gestational age, About 50% of the hypoglycemic babies may remain asymptomatic. The factor mainly protective against fetal hypoglycemia is the optimal control of maternal hyperglycemia especially during the third trimester and during labour. It had been shown that a mean maternal plasma glucose>105mg/dl during the last four hours of labour in a diabetic mother leads to a higher incidence of neonatal hypoglycemia.
- About 25% of the infant of diabetic mothers may present with serum calcium of <7 mg/ dl and this may remain mostly asymptomatic and is usually detectable during the 2nd and 3rd day of birth, Hypomagnesemia may coexist and may require correction.
- Respiratory distress syndrome (RDS) occurs in about 5% of the infant of diabetic mothers and it is seen equally in gestational diabetes mellitus. Against a strict glycaemic control reduces the incidence of RDS.
- Polycythemia is relatively common in infant of diabetic mother. The hyperviscosity due to polycythemia may induce congestive heart failure and vascular thrombosis accounting for the increased risk of renal vein thrombosis in these infants.
- Hyperbilirubinemia, the common abnormality is due to increased bilirubin production and decreased life span of the RBCs with glycosylated cell membranes. Hepatic conjugation of bilirubin may be impaired due to an immature liver.

Gestational Diabetes Mellitus is mostly the forerunner of Type-2 Diabetes Mellitus(9). Like type-2 Diabetes mellitus, obesity and advanced maternal age increase the risk of gestational diabetes mellitus(10,11).

PREVALENCE

Ethnically Indian Women are more prone to develop glucose intolerance during pregnancy and have eleven fold increased risk compared to white Caucasian necessitating universal screening during pregnancy(7).

Prevalence of Gestational Diabetes is 2% to 5% of all pregnancies in the United States(10). Marked variation has been reported in the prevalence of gestational diabetes world wide. The frequency of gestational diabetes mellitus ranged widely from 0.15% to as high as 50% in pima Indians in the USA (13).

MANAGEMENT

A very large number of these pregnant diabetics progress through pregnancy to manifest the consequences near or at term,when not much of effective intervention can be done. In those detected to have GDM and are not controlled with medical nutrition therapy,we have to start them on insulin, a potential drug with its attendant problems.Patient compliance is a problem with insulin as it has to be given subcutaneously and is painful. Not all patients are able to self administer the drug. In addition there are the complications such as hypoglycemia.

Pregnancy in individuals with diabetes requires meticulous planning and adherence to strict treatment regimes.Intensive diabetes management and normalisation

of the HbA1c are the standard of care for pregnant diabetics. The most crucial period of glycemic control is soon after fertilisation. The risk of fetal malformations is increased 4 to 10 times in individuals with uncontrolled diabetes at the time of conception and normal plasma glucose during the preconceptional period and throughout the periods of organogenesis should be maintained(6). Fasting and post prandial blood glucose not more than 90 and 120 mg% are targeted. Optimum fetal outcome is with values around 105mg%. Treatment options now used are

- medical nutrition therapy
- insulin
- glyburide & metformin

Maintaining glucose levels within target range requires meticulous attention to diet and physical activity. For many patients, monitoring capillary glucose several times daily and injecting insulin frequently is impossible. For this reason, there are many current initiatives to augment glucose control with oral agents, particularly in patients with insulin resistant type 2 diabetes. An ideal treatment would reduce insulin resistance, improve insulin secretion or action, and delay uptake of glucose from gut. Current strategies are aimed at augmentation of insulin supply [sulphonylureas & insulin therapy], amelioration of insulin resistance [exercise, weight loss, metformin & troglitazone therapy], prevention of fetal complications [maintenance of euglycemia].

α-GLUCOSIDASE INHIBITORS

- **Acarbose, voglibose**
- **Miglitol**

Acarbose:

Originally developed in Germany, is a pseudo-tetrasaccharide derived from the fermentation process of the fungus Actinoplanes utahensis. It is a class of oral agents that reversibly inhibit pancreatic amylase and α-glucosidase enzymes in the small intestines, delaying cleavage of complex sugars to monosaccarides and thus reducing the rise of blood glucose after a meal. Although these agents offer particular promise in pregnant women because of limited uptake from the gut,few studies in pregnancy are available to assess efficacy. α-glycosidase inhibitors reduce postprandial hyperglycemia by delaying glucose absorption; they do not affect glucose utilisation or insulin secretion. Postprandial hyperglyceamia, secondary to impaired hepatic and peripheral glucose disposal, contributes significantly to the hyperglycemic state in GDM. These drugs, taken just before each meal, reduces glucose absorption by inhibiting the enzyme that cleaves oligosaccharides into simple sugars in the intestinal lumen.

This class of agents is not as potent as other oral agents in lowering the hemoglobin A1c but it is unique because it reduces the postprandial glucose rise even in individuals with type 1 DM. The average decrease in post prandial blood glucose during Acarbose treatment in diet treated type 2diabetic patients was 3mmol/l and maximal decrease in HbA1c was 1%(29). If hypoglycemia occurs while taking these agents, the patient should consume glucose since the degradation and absorption of complex carbohydrates will be retarded.(6) Miglitol is absorbed systemically, hence not used in pregnancy.

Pharmacokinetics:

Given orally less than 2% is absorbed as the oral drug. It is metabolized in the GI tract primarily by the intestinal bacteria and to a lesser degree by the digestive enzymes. Urine contains 2% of the drug and its metabolites. As a result, accumulation of the drug does not occur when given thrice daily.

Adverse effects:

Flatulence, soft stools, diarrhoea, abdominal distention and pain, rarely abnormal liver function tests and skin reaction. The major side effects (diarrhoea, flatulence, abdominal distention) are related to increased delivery of oligosaccharides to the large bowel and can be reduced somewhat by gradual upward dose titration.

Contraindications:

Ulcerative colitis, partial intestinal obstruction, hepatic impairment, severe renal impairment, hernia, history of abdominal surgery. Simultaneous treatment with antacids is to be avoided. These agents should not be used in individuals with inflammatory bowel disease, gastroparesis, or serum creatinine >2.0mg/dl

Interactions:

Sucrose: Abdominal discomfort and diarrhoea.

Digestive enzymes [pancreatic amylase] and intestinal adsorbents: reduce efficacy of Acarbose.

Sulphonyl ureas, biguanides: Acarbose increases their efficacy.

Insulin: In IDDM, insulin requirement is reduced.

Dosage:

Therapy should be initiated at a low dose [25mg] with the evening meal and may be increased to a maximal dose over weeks [50-100mg Acarbose or 50mg Miglitol with each meal].

IN PREGNANCY-Category B

These drugs act within the intestinal lumen and are not absorbed systemically. They thus have no adverse effect on the developing fetus. Few breakdown products which may enter the circulation do not have any known teratogenic effect. Reproduction studies has been done in rats at doses upto 480mg/kg (corresponding to 9 times the exposure in humans, based on drug blood levels) and have revealed no evidence of impaired fertility or harm to the fetus due to Acarbose. In rabbits, reduced maternal weight gain, probably the result of the pharmacodynamic effect of high doses of Acarbose in the intestines, may have been responsible for a slight increase in the number of embryonic losses. However, rabbits given 160mg/kg Acarbose (corresponding to 10 times the dose in man, based on body surface area) showed no evidence of embryotoxicity and there was no evidence of teratogenicity at a dose 32 times the dose in man.(27)

De Veciana et al, 2002 reported in abstract a randomized trial of acarbose versus insulin in 110 patents with GDM. Glycaemic control was considered equivalent in the 2groups, although 6% of the acarbose group required insulin treatment because of gastrointestinal side effects. No significant adverse reactions or fetal anomalies

occurred. Acarbose is given prior to meals, initially 25mg orally 3 times daily, to a maximum of 100mg orally 3 times daily. Both groups improved their glucose levels with treatment. There was no differences between groups in maternal demographics, duration of therapy, glucose levels, birth weight, gestational age, or ceasarian section rate, she said (28).

Acarbose is classified by the **Food and Drug Administration** in pregnancy risk **category B,** meaning there is no evidence that it poses a risk to human fetuses.

AIMS AND OBJECTIVES

1To study Acarbose as a new line of management in GDM.

2Study its efficacy in glycaemic control, prevention of maternal complications & fetal outcome.

MATERIALS AND METHODS

1. Study design : Prospective study.

2. Study place : Institute of Obstetrics & Gynaecology, Chennai.

3. Study population : Pregnant GDM between 16-32

weeks gestation.

4. **Sample size** : 30

5. **Year of study** : January 2007- July 2008

INCLUSION CRITERIA

1. GDM with 75g GTT >140mg % at 2 hours.
2. Patients whose blood sugar was not controlled with medical nutrition therapy, i.e: FBS>95mg/dl or PPBS>120mg/dl.
3. 16-32 weeks of gestation.

EXCLUSION CRITERIA

1< 16 wk, > 32 weeks gestation.

2Known Diabetic.

3If blood sugar is controlled with medical nutrition therapy. i.e; FBS<95mg/dl or PPBS<120mg/dl.

4Multiple pregnancy.

5Anomalous fetus.

METHOD OF THE STUDY

Pregnant women of gestational age between 16 to 32 weeks attending the out patient department for their antenatal check up were subjected to the WHO 75 gm glucose challenge test. Known diabetics were excluded. All those who had a blood glucose value ≥ 140mg/dl at 2 hours were started on medical nutrition therapy for 2 weeks.

After 2 weeks, patients were instructed to come in an over night fasting state of 8 hours. A 2 hour glucose tolerance test was done and only those patients who had a fasting blood glucose >95mg/dl and/or 2 hour post prandial blood glucose >120mg/dl were included in the study after obtaining an informed consent.

All patients included in the study were started on Acarbose, the minimum dose being 25 mg BD. At the outset baseline liver function test, lipid profile and maternal HbA1c was obtained. After starting the patient on Acarbose they were reviewed every 15 days with fasting and postprandial blood sugar measurement alternating with glycemic profile every fortnight. The drug dose was titrated depending on the response and blood glucose levels to achieve optimum control. The aim was to have a mean blood glucose of 105 mg/dl. If blood sugar was not controlled with Acarbose they would be switched over to insulin therapy. Ultrasound for anomalies, to monitor growth, detect any deviation from the normal growth curve, amount of liquor and for fetal well being was done at 20-22 weeks, 28 weeks,32 weeks and at 36 weeks. The patients were thus followed until delivery. Immediately after delivery the maternal blood sugar and HbA1c

was measured to assess the glycaemic control that was achieved with the drug. The cord blood insulin levels were also measured to look for hyperinsulinism in the newborn baby. Perinatal outcome was recorded. All blood sugar measurements were done using the Enzymatic method in our hospital.

RESULTS AND ANALYSIS

TABLE - 1

AGE

AGE	FREQUENCY	PERCENTAGE
20-25 yr	14	46.7
26-30 yr	12	86.7
31-35 yr	3	10.0
36-40 yr	1	3.3
Total	**30**	**100**

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
AGE	30	26.5667	4.35243	.79464

The mean age of GDM patients was 26.5667 years and majority of the cases were in the <25 years group

Table - 2
Gestational Age

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
SCREENING GCT	30	162.2000	22.76931	4.15709
INCLUSION GTT	30	144.7050	24.87467	4.54147
INCLUSION GTT GROUP	30	3.0667	2.51798	.45972

The mean Gestational age at Screening is 26.00 weeks ,at Inclusion is 27.933 and at Delivery is 38.433.

Table - 3
Obstetric history

GRAVIDITY	FREQUENCY	PERCENTAGE
Primi	9	30.0
Gravida2	15	50.0
Gravida3	5	16.7
Gravida4	1	3.3

30% of the GDM cases were Primis and the incidence of GDM was more in multi gravida than in Primis.

Table - 4

Family history

FAMILY HISTORY	FREQUENCY	PERCENTAGE
Present	22	73.3
Absent	8	26.7
Total	**30**	**100**

73.3% of the cases of GDM had a positive family history of Diabetes.

Table - 5

BMI group

BMI CATEGORY	FREQUENCY	PERCENTAGE
<18.5	8	26.7
18.5-24.9	18	60.0
25-29.9	3	10.0
>30	1	3.3
Total	**30**	**100**

Majority of the cases of GDM(60%) fell in the normal BMI group, though 7 of them(26.7%) had BMI less than normal.

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
BMI	30	21.8117	3.60667	.65849

The mean BMI value among the GDM cases was21.8117 which fell in the normal BMI range.

BMI GROUP * MAXIMUM DOSE GROUP Crosstabulation

			MAXIMUM DOSE GROUP					
			1.00	2.00	3.00	4.00	6.00	7.00
BMI GROUP	1.00	Count	3	1	2	2	0	0
		% within MAXIMUM DOSE GROUP	37.5%	20.0%	40.0%	28.6%	.0%	.0%
		% of Total	10.0%	3.3%	6.7%	6.7%	.0%	.0%
	2.00	Count	4	3	3	4	1	0
		% within MAXIMUM DOSE GROUP	50.0%	60.0%	60.0%	57.1%	100.0%	.0%
		% of Total	13.3%	10.0%	10.0%	13.3%	3.3%	.0%
	3.00	Count	0	1	0	1	0	1
		% within MAXIMUM DOSE GROUP	.0%	20.0%	.0%	14.3%	.0%	100.0%
		% of Total	.0%	3.3%	.0%	3.3%	.0%	3.3%
	4.00	Count	1	0	0	0	0	0
		% within MAXIMUM DOSE GROUP	12.5%	.0%	.0%	.0%	.0%	.0%
		% of Total	3.3%	.0%	.0%	.0%	.0%	.0%
Total		Count	8	5	5	7	1	1
		% within MAXIMUM DOSE GROUP	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
		% of Total	26.7%	16.7%	16.7%	23.3%	3.3%	3.3%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	17.100[a]	18	.516
Likelihood Ratio	14.954	18	.665
Linear-by-Linear Association	.468	1	.494
N of Valid Cases	30		

The BMI did not correlate with the titration of drug dosage, they were found to be independent of each other.($p>0.05$ not significant).

Table - 6

Delivery mode

	FREQUENCY	PERCENTAGE
Labour natural	12	40.0
LSCS	5	16.7
Total	**17**	**56.7**

17 patients had previous pregnancies carried to term, 12 (70.6%)had a normal delivery while 5(29.4%) of them had a Caesarean section.

Table - 7

Indication for Caesarean section:

INDICATION	FREQUENCY	PERCENTAGE
Fetal distress	2	6.7
PROM with failed acceleration	1	3.3
CPD	1	3.3
Placenta preavia	1	3.3

Table - 8

Maternal complications

	Past pregnancy	**Present pregnancy**
PIH	3	5
PCOS	1	2
IGT	1	0

In the preceding pregnancy the most common maternal complication was PIH, which is a risk factor in the present pregnancy too.

Table - 9

Fetal wastage in previous pregnancies:

NUMBER OF ABORTIONS	FREQUENCY	PERCENTAGE
1	7	23.3
2	1	3.3

In the study group 8 patients had previous early pregnancy losses,1 of whom had 2 previous abortions.

Neonatal complications in previous pregnancies:

COMPLICATION	FREQUENCY	PERCENT
IUD	1	3.3
Neonatal jaundice	1	3.3

In the study group 1 baby died in the perinatal period of Jaundice and one patient had an intra uterine fetal demise.

Table - 10
Glycaemic control:

Blood sugar at screening:

BLOOD SUGAR(mg%)	FREQUENCY	PERCENTAGE
140-149	11	36.3
150-159	6	20
160-169	7	23.3
170-179	2	6.7
180-189	0	0
190-199	1	3.3
200-209	0	0
210-219	2	6.7
220-229	0	0
230-239	1	3.3

Table - 11
Mean Blood sugar at inclusion

BLOOD SUGAR (mg %)	FREQUENCY	PERCENTAGE
120-129	6	20
130-139	10	33.3
140-149	8	26.7
150-159	2	6.7
160-169	0	0
170-179	2	6.7
180-189	0	0
190-199	0	0
200-209	0	0
210-219	1	3.3
220-229	0	0
230-239	1	3.3

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
SCREENING GCT	30	162.2000	22.76931	4.15709
INCLUSION GTT	30	144.7050	24.87467	4.54147
INCLUSION GTT GROUP	30	3.0667	2.51798	.45972

The mean Blood sugar at screening is162.20 and at inclusion is 144.7050,which reflects that the blood sugar is uncontrolled even after medical nutrition therapy.

Table - 12

DRUG DOSAGE

DRUG DOSE(mg)	FREQUENCY	PERCENTAGE
75	8	26.7
100	5	16.7
125	5	16.7
150	6	20.0
175	1	3.3
200	1	3.3
225	1	3.3
250	3	10.0
Total	**30**	**100.0**

The mean dosage of the drug used was 131.6667mg with S.D.55.29441. 26.7% of the patients required a maximum of 75mg only, the next largest group23.3% requiring 150 mg. The maximum dose used was 250 mg which was used in 10% of the cases.

PRESENT BIRTH WEIGHT Vs MAXIMUM DOSE Cross tabulation

			MAXIMUM DOSE GROUP					
			1.00	2.00	3.00	4.00	6.00	7.00
PRESENT BIRTH WEIGHT GROUP	1.00	Count	1	0	0	0	0	1
		% of Total	3.3%	.0%	.0%	.0%	.0%	3.3%
	2.00	Count	1	0	1	0	0	0
		% of Total	3.3%	.0%	3.3%	.0%	.0%	.0%
	3.00	Count	3	1	1	3	0	0
		% of Total	10.0%	3.3%	3.3%	10.0%	.0%	.0%
	4.00	Count	2	2	1	3	0	0
		% of Total	6.7%	6.7%	3.3%	10.0%	.0%	.0%
	5.00	Count	1	0	1	0	0	0
		% of Total	3.3%	.0%	3.3%	.0%	.0%	.0%
	6.00	Count	0	1	1	0	0	0
		% of Total	.0%	3.3%	3.3%	.0%	.0%	.0%
	7.00	Count	0	1	0	0	1	0
		% of Total	.0%	3.3%	.0%	.0%	3.3%	.0%
	9.00	Count	0	0	0	1	0	0
		% of Total	.0%	.0%	.0%	3.3%	.0%	.0%
Total		Count	8	5	5	7	1	1
		% of Total	26.7%	16.7%	16.7%	23.3%	3.3%	3.3%

One baby (3.3%) had a birth weight >4kg (macrosomia) but the maternal blood sugar was controlled with 150 mg of Acarbose.30% of the babies born had a birth weight ranging between 2.76-3.00 and 26.7% had birth weight between 2.51-2.75 all of whose maternal blood sugar was controlled with drug dosage not exceeding150 mg.

Table - 13

HbA1c-at inclusion and delivery

HbA1c (%)	AT INCLUSION	AT DELIVERY
4.5-5.0	0	1
5.0-5.5	1	2
5.5-6.0	6	18
6.0-6.5	7	7
6.5-7.0	13	1
7.0-7.5	2	0
7.5-8.0	1	1

A. AT INCLUSION

	N	Mean	Std. Deviation	Std. Error Mean
INCLUSION Hb1ac	30	6.4900	.54034	.09865

HbA1c values at inclusion into the study had a mean value 6.49% and S.D.0.54034

B. AT DELIVERY

	N	Mean	Std. Deviation	Std. Error Mean
MOTHER Hba1c	30	5.9767	.46660	.08519

HbA1c values at delivery, after treatment with Acarbose had a mean value of 5.9767% and S.D.0.46660

HbA1c at Delivery

HbA1c (%)	**FREQUENCY**	**PERCENTAGE**
4.5-5.0	1	3.3
5.0-5.5	2	6.7
5.5-6.0	18	60.0
6.0-6.5	7	23.3
6.5-7.0	1	3.3
7.0-7.5	0	0
7.5-8.0	1	3.3

At the time of delivery 60% of case had HbA1c between 5.5-6.0% and 23.3% between 6.0-6.5. Only 1 case (3.3%) had HbA1c >7.5%.

Table - 14

INCLUSION vs. DELIVERY HbA1c

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	INCLUSION Hb1ac	6.4900	30	.54034	.09865
	MOTHER Hba1c	5.9767	30	.46660	.08519

	Paired Differences			
	Mean	**Std. Deviation**	**Std. Error Mean**	**Sig. (2 - tailed)**
Inclusion HbA1c and Delivery HbA1c	.5133	.38303	.6993	.000

Comparing the maternal HbA1c at inclusion into the study and at the time of delivery has p=0.000 which is highly significant (Mc Nemar test)

Table - 15

MEAN GLYCEAMIC CONTROL:

Mean Blood Sugar

	N	Mean	Std. Deviation	Std. Error
.00	24	103.8021	6.76138	1.38016
1.00	1	115.3700	.	.
2.00	3	113.9067	6.94713	4.01093
4.00	1	103.0600	.	.
5.00	1	105.2100	.	.
Total	30	105.2203	7.26426	1.32627

The mean Blood sugar achieved during the antenatal period by titrating the drug dose is 105.2203mg/dl with S.D.7.26426

Inclusion GTT blood sugar Vs. Mean Blood sugar during Pregnancy:

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	INCLUSION GTT	144.7050	30	24.87467	4.54147
	MEAN BS	105.2203	30	7.26426	1.32627

		Paired Differences		
		Mean	Std. Deviation	Std. Error Mean
Pair 1	INCLUSION GTT - MEAN BS	39.4847	23.35831	4.26463

Significance (2-tailed) - .000. The mean blood sugar at inclusion is 144.7050mg/dl and during the antenatal period is 105.2203. The calculated value of p=0.000 which is highly significant.

Table - 16
Neonatal blood sugar:

BLOOD SUGAR (mg%)	FREQUENCY	PERCENTAGE
51-55	2	6.7
55-60	5	16.7
60-65	6	20.0
65-70	6	20.0
70-75	4	13.3
75-80	5	16.7
80-85	1	3.3
85-90	1	3.3
Total	30	100.0

Major proportion(40%) of the babies had blood sugar in the range of 61-70 mg/dl and only 2 (6.7%)babies with blood sugar between 51-55mg/dl.

	N	Mean	Std. Deviation	Std. Error Mean
BABY BS GROUP	30	3.9667	1.79046	.32689

The mean Neonatal blood sugar was 68.062435 mg/dl.

Table - 17
Birth weight

BIRTH WEIGHT (KG)	FREQUENCY	PERCENTAGE
2.0-2.25	2	6.7
2.25-2.5	3	10.0
2.5-2.75	8	26.7
2.75-3.0	9	30.0
3.0-3.25	3	10.0
3.25-3.5	2	6.7
3.5-3.75	2	6.7
3.75-4.0	0	0
4.0-4.25	1	3.3

	N	Mean	Std. Deviation	Std. Error Mean
PRESENT BIRTH WEIGHT	30	2.9000	.44856	.08190

The mean Birth weight of the babies born was 2.900 kg with S.D. 0.44856.

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	Mean	N	Std. Deviation	Std. Error Mean
PRE. BIRTH WEIGHT	2.6000	17	.60699	.14722
PRESENT BIRTH WEIGHT	2.9088	17	.50782	.12317

.

	Paired Differences		
	Mean	Std. Deviation	Std. Error Mean
PRE. BIRTH WEIGHT - PRESENT BIRTH WEIGHT	-.3088	.50132	.12159

The mean Birth weight of babies born in previous pregnancies (before treatment) was 2.600kg and in the present pregnancy is 2.9088kg. P=0.022 Significant.

Table - 18

Cord blood insulin

CORD BLOOD INSULIN(µg%)	FREQUENCY	PERCENTAGE
5.6-6.0	3	10.0
6.1-6.5	12	40.0
6.6-7.0	8	26.7
7.1-7.5	6	20.0
7.6-8.0	1	3.3

40% of the study group had Cord blood insulin of 6.1-6.5µg/dl and in 3.3% it was 7.6-8.0µg/dl

	N	Mean	Std. Deviation	Std. Error Mean
CORD BLOOD INSULIN	30	6.6033	.52751	.09631

Mean cord blood insulin of the babies delivered was 6.6033 µg/dlwith S.D.0.52751.

TABLE - 19

Ultrasound monitoring

USG at 22 weeks-1 case of Anomaly

USG at 28 weeks

USG FINDING	FREQUENCY	PERCENT
1.Macrosomia	1	3.3
2.Hydramnios	1	3.3

USG at 28 week revealed 1 case with Macrosomia and 1 case with Hydramnios.

USG at 32 weeks

USG FINDING	FREQUENCY	PERCENTAGE
1.Macrosomia	1	3.3
2.Hydramnios	1	3.3
3.Oligohydramnios	1	3.3

USG at 32 weeks revealed 1 case of Macrosomia, Hydramnios and IUGR each.

USG at 36 weeks

USG FINDING	FREQUENCY	PERCENT
1.Macrosomia	1	3.3
2.Hydramnios	3	10.0
3.Oligohydramnios	1	3.3
4.IUGR	1	3.3

USG at 36 weeks revealed 1 case of Macrosomia, 3 cases of Hydramnios, 2 cases of Oligohydramnios and 1 case of IUGR.

Table - 20

PRESENT DELIVERY

Delivery mode

MODE OF DELIVERY	FREQUENCY	PERCENTAGE
Labour natural	20	33.3
LSCS	10	66.7
Total	**30**	**100**

66.7% of the cases delivered normally and 33.3% were delivered by LSCS.

LIVE BABY

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1.00	30	100.0	100.0	100.0

100% of the patients delivered a live viable baby.

SEX:

SEX	FREQUENCY	PERCENTAGE
Male	15	50.0
Female	15	50.0

The babies delivered were 50% male and 50% female.

TABLE -21

APGAR

APGAR	FREQUENCY	PERCENTAGE
>7	29	96.7
<7	1	3.3

96.7% of the babies had a good Apgar score and 3.3% had an Apgar <7.

TABLE - 21

MATERNAL DELIVERY BLOOD SUGAR:

Inclusion GTT Vs Blood Sugar at Delivery

	Mean	N	Std. Deviation	Std. Error Mean
INCLUSION GTT	144.7050	30	24.87467	4.54147
BS DEL	84.8667	30	9.89857	1.80722

The mean Blood sugar at inclusion was 144.7050mg/dl and at delivery was 84.8667mg/dl.

		Paired Differences		
		Mean	Std. Deviation	Std. Error Mean
Pair 1	INCLUSION GTT - BS DEL	59.8383	25.34000	4.62643

P=0.000 which is highly significant.

DISCUSSION

A prospective study was conducted in IOG during Jan 2007-July 2008. For this study 1000 randomly selected pregnant women in 16-32 weeks gestation were screened and 30 cases of GDM were diagnosed based on WHO-GCT. They were included in the study.

PREVALENCE:

According to **Beischer et al, 1991,** the prevalence of GDM in the Indian subcontinent is 15%.

In a study conducted in Jean Verdier Hospital, France it was identified that prevalence of GDM was 15.65%, if universal screening is adopted.

In a study conducted by **Dr.Anjalakshi** for evaluation of diagnostic criteria for AGT in south Indian pregnant women, prevalence of GDM was found to be 15%.

Seshiah et al found the prevalence to be 16.2% in India in his study using WHO criteria.

Schmidt et al in 2001 found the incidence to be 7.2% by WHO criteria.

In our study the prevalence of GDM by WHO criteria was 3% since we were taking only women 16-32 weeks of gestation and excluding diabetics, multiple pregnancies and anomalous fetuses

AGE:

Coustan found that the incidence of GDM is 3.8% in women of 30-34 years of age but only 0.7% in women younger than 21 years in a larger study of unselected population.

Seshiah et al in his study in 2005 found that the incidence of DM in <20 years is 14.5%,20-24 years is 13.7%,25-29 years is 19.5%, ≥30 years is 25%.

In our study the mean age of the GDM patients was 26.5667 years.

GESTATIONAL AGE:

A study was done by **Seshiah et al** for detection of GDM in the three trimesters of pregnancy. Among the studied patients 16.3% were within 16 weeks of gestation, 23.1% were between 17-23 weeks of gestation,60.6% were more than 24 weeks .

In our study, the mean gestational age at screening was 26 weeks and at delivery was 38.433 weeks.

OBSTETRIC HISTORY:

Prevalence of gestational diabetes increases with gravidity from 16-3% in primis to 25-8% in gravida ≥ 4 in a study by **Seshiah et al**.

Pyke De et al found that the incidence of GDM increases with parity.

In our study, 30% of the DM cases were primis and the incidence of GDM was more in multi gravida than primis.

FAMILY HISTORY:

Serirat et al in 1992 have shown that family history of diabetes is present in 23.1% of patients with abnormal glucose tolerance.

Moses et al has shown that family history of diabetes is present in 11.6% of patients with GDM.

In our study, positive family history was present in 73.3% of cases of GDM (p=0.0).

BMI:

Serirat et al in 1992, in a study found that obesity was present in 26.5% of patients with GDM.

In a study by **Seshiah et al** the incidence of GDM was 33.3% in patients with BMI ≥ 30.

In our study 60% of cases of GDM fell in the normal BMI group, 26.7% had less than normal BMI and 3.3% had BMI ≥ 30. This shows that the incidence of GDM among the patients with low BMI should not be overlooked. In this study, the drug

dosage required to achieve glycemic control did not correlate with the BMI (p=0.516).

PREVIOUS DELIVERY:

17 cases had previous deliveries of which 70.6% was normal delivery and 29.4% had Caesarean section. In the study 66.7% had normal delivery and 33.3% had Caesarean section. The difference between the previous delivery when they did not have GDM and now when they do is not significant. there were no difficult deliveries either.

MATERNAL COMPLICATIONS

Maternal mortality has become rare in women with diabetes a emphasized by **Cousins** who stated that mortality is increased 10 fold, most often as a result if ketoacidosis, hypertension, preeclampsia, pyelonephritis and patients with coronary artery disease. In our study there was no morality.

Suhoven and Terano et al in 1993 reported the incidence of PIH and preeclampsia to be 2 times more common among GDM patients than controls (19.8% vs. 10%).

In a study **Schmidt et al**, it was found that frequency of preeclampsia was 5%.

PIH was seen in 13.7% in GDM in a study by **Cousins et al.**

In our study, PIH in the preceding pregnancy was the most common risk factor and in the present pregnancy complicated by GDM, the incidence of PIH is shown to be more.

FETAL WASTAGE:

Zarate et al in 2000, did a study in 6 pregnant women who were treated with

Acarbose. All pregnancies were uneventful and the newborns were considered normal.

Sherry Boschert in Aug,2002 compared insulin therapy to Acarbose treatment in GDM. She found no significant difference between the two groups in fetal outcome.

In the study group 8 patients had previous early pregnancy losses,1 of whom had 2 previous abortions,1 patient had an IUFD and 1 patient lost the previous baby to perinatal jaundice. Thus the incidence of fetal wastage was 33.3%. After treatment with Acarbose all the present pregnancies were carried to term with no fetal wastage.96.7% of the babies had a good Apgar score of >7.

FETAL COMPLICATIONS:

Bertini et al, J.Perinatal medicine, 2005 studied the perinatal outcome in GDM cases managed with insulin, glyburide and Acarbose. The rate of large for gestational age fetuses in each group was 3.7%, 25% and 10.5% respectively.

At term, in our study, there was 1 case of macrosomia-3.3% (with onset before the start of treatment with Acarbose), 3 cases of hydramnios 10%, 2 cases of oligohydramnios and 1 case of IUGR which was most probably due to the accompanying PIH in that mother.

GLYCAEMIC CONTROL:

In studies conducted **Margarita deVeciana, ACOG** showed glycaemic control similar to insulin was achieved, with only 5% who had to be switched over to insulin due to GI side effects.

Bertini AM et al found that glucose control was not achieved in 42.1% of

patients using Acarbose.

In our study, Patients were started on Acarbose and the glycaemic control was good with the mean blood sugar being 105.3303mg/dl. (p-0.00). No patient had hyperglyceamia which required change of treatment.

At the time of delivery the mean blood sugar was 84.8667mg/dl indicating good control of blood sugar is achieved using Acarbose alone (p=0.00).

DRUG DOSAGE:

Margarita de Veciana in her study with 56 patients in the Acarbose group started out taking 25mg of the agent thrice daily with the first bite of each meal. The dose was increased as needed to a maximum of 100mg TDS. At term, the mean total daily dosage was 120mg/day in the Acarbose group.

This is comparable to our study where the mean total daily dose was 131.667mg/day. The maximum dose used in our study was 250mg in 10% of the cases, while most cases achieved glycemic control with 75mg (26.7%) and 150mg (23.3%).

HbA1c:

De Veciana et al in their study showed that the HbA1c reduction with Acarbose was comparable to insulin.

VS Reddy, RK Sahay et al in their paper on newer oral ant diabetic agents said the maximal decrease in HbA1c that could be achieved with Acarbose was 1%.

In our study the mean HbA1c when the patients were diagnosed to have GDM was 6.49%. After treatment with Acarbose the mean HbA1c at delivery was 5.9767% (p=0.00) showing a reduction of 0.52%.

NEONATAL BLOOD SUGAR:

Cowett et al found that infants of women with GDM have an incidence of neonatal hypoglycemia that approaches 30-50%.

According to **James** the frequency of hypoglycemia is 18-49%.

The mean neonatal blood sugar was 68.06mg/dl with 40% of babies having blood sugar 61-70mg/dl and only 6.7% with blood sugar between 51-55mg/dl in our study. There were no cases of hypoglycemia, indicating there was no hyperinsulinemia in the babies causing this complication.

BIRTH WEIGHT:

Spellacy, WN Miller, S Winegar found that Macrosomia was present in 50% of pregnancies with GDM.

Langer et al also found 50% of pregnant patients with GDM to have

Macrosomia.

In our study the mean birth weight was 2.9 kg with 1 case of macrosomia comparing with previous pregnancy birth weight of 2.6kg, there is a significant difference. But there was no resulting complications due to the increase in birth weight.

CORD BLOOD INSULIN:

In our study, the mean cord blood insulin was 6.6033µg/dl with 40% having cord blood insulin of 6.1-6.5µg/dl.

SUMMARY

1.In the patients recruited into the study, the mean age was 26.5667 years.

2.73.3% of GDM cases had a positive family history.

3.60% of GDM cases had a normal BMI.BMI did not correlate with titration of drug dosage which has to be individualized.

4.The maternal complications encountered in the study was PIH and PCOS ,the incidence of which was the same as non-GDM cases.

5.The fetal wastage in the previous pregnancies was 47.62% when the patients were not diagnosed or treated for GDM. In the study fetal complications were only 3.33%(1 case of respiratory distress) after treatment with Acarbose. Macrosomia and fetal hyperinsulinism were prevented by Acarbose.1 case of macrosomia had onset before Acarbose was started and hence was excluded from our statistics.

6.Mean drug dose needed for glycemic control was 131.6667mg with most patients requiring 75 -150mg of Acarbose. But the dosage has to be individually titrated for each patient.

7.Maternal HbA1c and maternal blood sugar was statistically reduced after treatment with Acarbose.

8.Glycemic control was good and the aimed level of 105mg% was achieved with Acarbose treatment.

9.The mean neonatal blood sugar was 68.062435mg/dl with no baby going for

hypoglycemia.

10. In the present pregnancy, all pregnancies were safely carried to term, 66.7% of patients delivered normally, with 96.7% of babies having a good Apgar score.

CONCLUSION

GDM patients treated with Acarbose had good glycemic control during the antenatal period and delivery. It prevented the maternal complications due to hyperglycemia, as well as complications for the fetus like macrosomia, anomalies, growth restriction, hyperinsulinism, hypoglycemia, etc. The fetal outcome after treatment of GDM with Acarbose was good. There were no adverse effects of the drug. It obviated the need for use of insulin.

Thus Acarbose is a safe and effective oral drug in the management of GDM achieving good glycaemic control and preventing maternal and fetal complications of GDM.

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PROFORMA:

A. GENERAL:

Name: Age: Education:

Address & telephone no:

Date of inclusion into study:

B. OBSTETRIC HISTORY:

LMP: EDD:

Gestational age at time of inclusion into study:

Gravida	Para	Abortion	Stillbirth

C.FAMILY HISTORY:

Father: 1st degree relatives:

Mother: Siblings:

D.GENERAL EXAMINATION:

Anemia:

Pedal edema:

Acanthosis nigricans:

B.P.

Ht: Wt:

Prepregnancy Wt:

BMI(calculated from prepregnancy wt.):

E. ASSOCIATED PROBLEMS IN PREVIOUS PREGNANCY:

Pregnancy	Yes/no Delivered	it wasted If no, was	IGT GDM/	PIH	PCOD	disorders Endocrine	Proteinuria	taken Treatment	information Other
1.									
2.									

3.									
4.									
5.									

F. PREVIOUS PRENANCY OUTCOME:

Pregnancy	Date of delivery	Labour natural	Assisted	Caesarian	Sex of baby	Birth weight	Congenital abnormalities	Other Complications
1.								
2.								
3.								
4.								
5.								

G. SCREENING DETAILS:

Date of screening :

Week of gestation :

FPG :

2 hour PPG :

H. OTHER INVESTIGATIONS:

Microalbuminuria :

Retinopathy :

Others: :

I. TREATMENT SCHEDULE:

Visit log	Date of visit	Gestational week	Plasma glucose	Acarbose dose

J. USG FINDINGS:

22 weeks	28 weeks	32 weeks	36 weeks

K. OUTCOME OF PRESENT PREGNANCY:

Delivered-yes/no

If no,was it wasted:

If delivered:

Date of delivery:

Sex of baby:

Mode of delivery:spontaneous( ) Induction( ) Caesarean( )

Weight:

Height:

Apgar :

New born infants blood glucose:

L. MATERNAL COMPLICATIONS:

Proteinuria:

Retinopathy:

Pre-eclampsia:

PCOD:

Others:

M. NEONATAL COMPLICATIONS:

Congenital abnormality (if yes, specify):

Neonatal hypoglycemia:

Shoulder dystocia:

Other information:

ABBREVIATIONS

IQ	-	Intelligence Quotient
RDS	-	Respiratory Distress Syndrome
USA	-	United States of America
GDM	-	Gestational Diabetes Mellitus
DM	-	Diabetes Mellitus
WHO	-	World Health Organisation
BD	-	Bi Diurnal
HbA1c	-	Heamoglobin A1c
BMI	-	Body Mass Index
LSCS	-	Lower Segment Caesarean Section
PROM	-	Premature Rupture Of Membranes
CPD	-	Cephalo Pelvic Disproportion
PIH	-	Pregnancy Induced Hypertension
PCOS	-	Poly Cystic Ovarian Syndrome
IGT	-	Impaired Glucose Tolerance
IUD	-	Intra Uterine Death
USG	-	Ultrasonogram
LMP	-	Last Menstrual Period
EDD	-	Expected Date Of Delivery

KEY TO MASTER CHART

Obstetrics History:

Gravida 1 - 1

Gravida 2 - 2

Gravida 3 - 3

Family History:

Present - +

Absent - –

Delivery Mode:

Labour Natural - 1

Caesarian Section - 2

Maternal complications:

PIH - 1

PCOD - 2

IGT - 3

Fetal Wastage:

One abortion - 1

Two abortion - 2

Three abortion - 3

Glycemic control:

Good - G

Fair - F

Poor - P

Ultrasound :

Normal	-	N
Hydramnios	-	Hyd
Oligohydramnios	-	Oligo
Macrosomia	-	Macro
Renal Anomaly	-	Renal An.

Live Baby:

Live Baby - 1

Dead Born - 2

Sex :

Male - M

Female - F

Apgar score:

More than 7 at 10 minutes - G

Less than 7 at 10 minutes - F

Less than 5 at 10 minutes - P